U.S. Department of Labor

Office of Administrative Law Judges
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Issue Date: 06 April 2004

Case No. 1999-BLA-1036

In the Matter of:
GERALDINE THACKER, Widow of
ZACK THACKER, Miner
Claimant,

v.

SCOTTS BRANCH COAL COMPANY,
Employer,
and
MAPCO, INC.,
Carrier,

and
DIRECTOR, OFFICE OF WORKERS'
COMPESATION PROGRAMS.
Party-in-Interest.

APPEARNCES:
Thomas G. Polites, Esq.
On behalf of Claimant

Laura Metcoff Klaus, Esq.
On behalf of Employer/Carrier

DECISION AND ORDER ON SECOND REMAND – DENIAL OF BENEFITS

This is a decision and order arising out of a claim for benefits under Title IV of the Federal Coal Mine Health and Safety Act of 1969, as amended by the Black Lung Benefits Act of 1977, 30 U.S.C. §§ 901-962, ("the Act") and the regulations thereunder, located in Title 20 of the Code of Federal Regulations. Regulation section numbers mentioned in this Decision and Order refer to sections of that Title.[1]

[1] The Department of Labor amended the regulations implementing the Federal Coal Mine Health and Safety Act of 1969, as amended. These regulations became effective on January 19, 2001, and are found at 65 Fed. Reg. 80, 045-80,107 (2000)(to be codified at 20 C.F.R. Parts 718, 722, 725 and 726). On August 9, 2001, the United States District Court for the District of Columbia issued a Memorandum and Order upholding the validity of the new regulations. All citations to the regulations, unless otherwise noted, refer to the amended regulations.

On June 25, 2003, this case was remanded to the Office of Administrative Law Judges by the Benefits Review Board for further consideration consistent with its decision and order.[2] On remand, all parties were permitted to file a brief.

ISSUES

The issues in this case are:

1. Whether the Miner had pneumoconiosis as defined by the Act;

2. Whether the Miner's pneumoconiosis arose out of coal mine employment; and

3. Whether the Miner's death was due to pneumoconiosis;

Based upon a thorough analysis of the entire record in this case, with due consideration accorded to the arguments of the parties, applicable statutory provisions, regulations, and relevant case law, I hereby make the following:

FINDINGS OF FACT AND CONCLUSIONS OF LAW

Procedural History

A thorough account of the procedural history has been set forth in the undersigned's decision and orders dated May 30, 2000 and May 21, 2002. After the undersigned issued a decision and order on remand denying survivor's benefits on May 21, 2002, Claimant sought review before the Benefits Review Board ("Board"). The Board issued a decision and order affirming in part, vacating in part, and remanding the claim to the undersigned for further consideration on June 30, 2003. The Board noted that it had previously affirmed the undersigned's finding that Claimant had established eleven years of coal mine employment. The Board vacated the undersigned's finding under § 718.202(a)(2), and instructed the undersigned to consider whether the evidence establishes the presence of pneumoconiosis under § 718.202(a)(1) and (a)(4) if the undersigned determines that the autopsy evidence is insufficient to establish the presence of pneumoconiosis. In light of its decision to vacate the undersigned's finding under § 718.202(a)(2), the Board vacated the undersigned's finding under § 718.205(c).

The undersigned issued an order on October 6, 2003, permitting the parties to file a brief on remand. Claimant and Employer both filed briefs on remand.

MEDICAL EVIDENCE

I incorporate the chest x-ray interpretations, pulmonary function tests, arterial blood gas studies, narrative medical reports, hospital records, biopsy, and autopsy evidence contained in the undersigned's decisions issued on May 30, 2000 and May 21, 2002, and the decision and

[2] In this Decision, "DX" refers to the Director's Exhibits, "EX" refers to the Employer's Exhibits, "CX" refers to the Claimant's Exhibits, and "Tr" refers to the official transcript of this proceeding.

order of Administrative Law Judge Lee Romero issued on October 2, 1992, to the extent that it is not inconsistent with the evidence summarized herein.

DISCUSSION AND APPLICABLE LAW

Mrs. Thacker filed her survivor's claim on June 8, 1998. Entitlement to benefits must be established under the regulatory criteria at Part 718. *See Neeley v. Director, OWCP*, 11 B.L.R. 1-85 (1988). The Act provides that benefits are provided to eligible survivors of a miner whose death was due to pneumoconiosis. § 718.205(a). In order to receive benefits, the claimant must prove that:

1). The miner had pneumoconiosis;

2). The miner's pneumoconiosis arose out of coal mine employment; and

3). The miner's death was due to pneumoconiosis.

§§ 718.205(a). Failure to establish any of these elements by a preponderance of the evidence precludes entitlement. *See Anderson v. Valley Camp of Utah, Inc.*, 12 B.L.R. 1-111, 1-112 (1989); *Trent v. Director, OWCP*, 11 B.L.R. 1-26, 1-27 (1987).

Pneumoconiosis

In establishing entitlement to benefits, Claimant must initially prove the existence of pneumoconiosis under § 718.202. Claimant has the burden of proving the existence of pneumoconiosis, as well as every element of entitlement, by a preponderance of the evidence. *See Director, OWCP v. Greenwich Collieries*, 512 U.S. 267 (1994). Pneumoconiosis is defined by the regulations:

> For the purpose of the Act, "pneumoconiosis" means a chronic dust disease of the lung and its sequelae, including respiratory and pulmonary impairments, arising out of coal mine employment. This definition includes both medical, or "clinical" pneumoconiosis and statutory, or "legal" pneumoconiosis.
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> (1) *Clinical Pneumoconiosis*. "Clinical pneumoconiosis" consists of those diseases recognized by the medical community as pneumoconiosis, i.e., conditions characterized by permanent deposition of substantial amounts of particulate matter in the lungs and the fibrotic reaction of the lung tissue to that deposition caused by dust exposure in coal mine employment. This definition includes, but is not limited to, coal workers' pneumoconiosis, anthracosilicosis, anthracosis, anthrosilicosis, massive pulmonary fibrosis, silicosis or silicotuberculosis, arising out of coal mine employment.
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> (2) *Legal Pneumoconiosis*. "Legal pneumoconiosis" includes any chronic lung disease or impairment and its sequelae arising out of coal mine employment. This

definition includes, but is not limited to, any chronic restrictive or obstructive pulmonary disease arising out of coal mine employment.

Section 718.201(a).

Section 718.202(a) sets forth four methods for determining the existence of pneumoconiosis.

(1) Under § 718.202(a)(1), a finding that pneumoconiosis exists may be based upon x-ray evidence. The record consists of 34 x-ray interpretations of 19 chest x-rays obtained between 1976 and 1992, as well as three more interpretations of three x-rays obtained in 1998. Of the 34 interpretations obtained between 1976 and 1992, only six were positive. Of the six positive interpretations, only two were rendered by a B-reader, and the other four were rendered by physicians with no advanced credentials for interpreting chest x-rays. To the contrary, 20 negative interpretations were rendered by physicians who were dually-certified as radiologists and B-readers. The chest x-ray evidence developed between 1976 and 1992 is overwhelmingly negative based on the more numerous negative interpretations rendered by better qualified physicians. The three x-ray interpretations of the three films obtained in the last two weeks before Miner's death were interpreted by Dr. Myers, who is a board-certified radiologist, as positive for the existence of pneumoconiosis. There were no contrary interpretations of the three films from 1998. I accord more probative weight to the three x-rays interpretations from 1998 because they are more probative of Miner's condition at the time of his death. Pneumoconiosis is recognized by the Act as a latent and progressive disease which may first become detectable after the cessation of coal mine employment. § 718.201(c). The possibility that Miner's pneumoconiosis may have been latent and progressive provides additional support for attributing greater weight to the more recent chest x-ray interpretations. Since it is possible for pneumoconiosis to be latent and progressive, and because the six year period between 1992 through 1998 is a significant amount of time, I find that Claimant has established the presence of pneumoconiosis by a preponderance of the chest x-ray evidence. Therefore, I find that the Claimant has established the existence of pneumoconiosis by x-ray evidence under subsection (a)(1).

(2) Under § 718.202(a)(2), a determination that pneumoconiosis is present may be based, in the case of a deceased miner, upon autopsy evidence. A report of an autopsy submitted in connection with a claim shall include a detailed gross macroscopic description and microscopic description of the lungs or visualized portion of the lung. § 718.106(a). A finding in an autopsy of anthracotic pigmentation shall not be sufficient, by itself, to establish the existence of pneumoconiosis. § 718.202(a)(2). Autopsy evidence is the most reliable evidence of the existence of pneumoconiosis. *Terlip v. Director, OWCP*, 8 B.L.R. 1-363 (1985). A diagnosis of pneumoconiosis issued by the autopsy prosector is entitled to significant probative value because the pathologist who performs the autopsy sees the entire respiratory system as well as other body systems. *See Fetterman v. Director, OWCP*, 7 B.L.R. 1-688, 1-691 (1985). It is proper to accord greater weight to the opinion of the autopsy prosector over the opinion of reviewing pathologists. *Peskie v. U.S. Steel Corp.*, 8 B.L.R. 1-126 (1985); *Similia v. Bethlehem Mines Corp.*, 7 B.L.R. 1-535 (1984). Additionally, it is reasonable to assign greater weight to physicians who have reviewed the autopsy slides over those physicians who have not. *See Terlip*

v. Director, OWCP, 8 B.L.R. 1-363 (1985). The Board has held that anthracosis found in lymph nodes may be sufficient to establish the existence of pneumoconiosis. *Taylor v. Director, OWCP*, BRB No. 01-0837 BLA (July 30, 2002) (unpublished). Six physicians rendered medical opinions after reviewing the autopsy slides.

Dr. Dennis conducted the autopsy of Miner on April 6, 1998. His report contains a macroscopic and microscopic description. The stated purpose of Dr. Dennis' autopsy was to determine the presence or absence of anthracosilicosis. He obtained sections from both of Claimant's lungs. In his macroscopic description, he noted that the pulmonary tissue is marked with black pigment deposition and emphysematous change with dilation of alveolar spaces. He also commented on the presence of black macules with pigment deposition varying in size from .3 to .5 cms. Dr. Dennis noted that sections through the pulmonary architecture show emphysematous changes, black pigment deposition, and black pigment surrounding major vessels and bronchi. Due to the adhesions, fibrosis and obliteration of the space in the pulmonary cavity, Dr. Dennis was only able to extricate a limited amount of tissue without tearing the lung tissue. He also stated that the lung sections are portions only because of the marked fibrosis present, which prevented complete removal. Under microscopic examination, Dr. Dennis detected the black pigment present showing birefringent crystalline structures compatible with silicosis. He found nodules varying in size and shape from .1 to .5 millimeters. He found that the arterioles show fibromuscular proliferation suggestive of cor pulmonale. Dr. Dennis commented that section P shows a marked proliferation of epitheloid histiocytes forming a nodule with some inflammation and histiocytic response along with markedly thickened pleura and fibrosis. In sections Q and R, he found markedly thickened pleura, silicotic and anthracotic nodules associated with collapse of the septal walls and subsequent collapse of alveolar septal spaces, and he also found broadened fibrous tissue septi along with granulomatous response. Dr. Dennis detected macules and papules. Regarding Miner's cardiovascular system, Dr. Dennis diagnosed left ventricular hypertrophy and biventricular hypertrophy. He found an adequate coronary artery system, and he referenced sections A and B. Under the heading of respiratory system, Dr. Dennis diagnosed: (1) pulmonary congestion, focal edema and restrictive pulmonary disease with marked pleural thickening, pleuritis, acute and chronic with marked anthracosilicotic pigment deposition forming macules and papules; (2) interstitial fibrosis with marked proliferation of fibrous connective tissue septi in alveolar walls with chronic inflammation; (3) granulomatous inflammation; and (4) anthracosilicotic pigment deposition and pleural reaction, moderate to severe with extreme pulmonary fibrosis and restrictive lung disease, with features related to anthracosilicosis. Dr. Dennis then found that Miner died a pulmonary death. He considered the pathology to be moderate to severe. He then stated: "[t]he etiological factors remain to be coal dust or anthracosilicotic pigment deposition with resultant fibrosis and pulmonary reactions secondary to this process."

I find that Dr. Dennis' opinion amounts to a diagnosis of clinical pneumoconiosis. He attributes the fibrosis and pulmonary reactions he detected to coal dust or anthracosilicotic pigment deposition. Dr. Dennis' autopsy report complies with the quality standards of § 718.106. He set forth pathological findings and observations, and his reasoning is supported by adequate data. His opinion is reasoned and documented. I find that Dr. Dennis' opinion is entitled to probative weight. Dr. Dennis was the autopsy prosector. He noted that he was only able to remove portions of lung sections because of the marked fibrosis that was present. At a

different point of his macroscopic description, Dr. Dennis stated that the adhesions, fibrosis, and obliteration of the space in the pulmonary cavity limited the amount of tissue that was available for extrication. The undersigned finds that it is reasonable to accord a greater degree of probative weight to the opinion of Dr. Dennis because his macroscopic examination of Miner's lung tissue provided him with a more complete picture of the nature and extent of the disease processes in Miner's lungs that reviewing pathologists were not privy to because Dr. Dennis was unable to prepare slides of the most damaged tissue due to the extent of the damage. Since Dr. Dennis' opinion is reasoned and documented, and because he was privy to a more complete picture of Miner's respiratory system, I accord an enhanced degree of probative weight to Dr. Dennis' opinion finding the presence of clinical pneumoconiosis.

Dr. Naeye rendered post-mortem and clinical findings in a narrative report dated August 8, 1998. He reviewed the autopsy report, death certificate, and 18 slides of tissue removed at autopsy. Dr. Naeye also reviewed medical records from various hospitals, as well as records from Drs. Hazlett and Nicholas. Dr. Naeye rendered microscopic findings from his review of the autopsy slides. He found that the dominating disorder was a granulomatous process that caused widespread destruction of normal lung tissue. Dr. Naeye commented that the age of this process varies, but he added that much of it was recent. He found that asbestos bodies and acute lobular pneumonia were absent. Dr. Naeye did find centrilobular emphysema varying in degrees of severity from mild to moderate. He did not detect any evidence of chronic bronchitis. Dr. Naeye stated that the amount of black pigment present in the lungs is small. At a few sites, Dr. Naeye detected some fibrous tissue associated with black pigment as well as rare admixed small and medium-sized birefringent crystals. He found it difficult to determine if any focal emphysema was present due to the interstitial granulomatous process. In his interpretation of the autopsy slides, Dr. Naeye stated that the minimal findings (a few small black deposits adjacent to small airways and arteries with some admixed fibrous tissue and a few birefringent crystals) required to make the diagnosis of simple coal workers' pneumoconiosis ("CWP") are present. Dr. Naeye diagnosed the presence of clinical pneumoconiosis based on his review and interpretation of the autopsy slides. He set forth pathological observations and findings, and his reasoning is supported by adequate data. His opinion is reasoned and documented. I find that Dr. Naeye's opinion is entitled to probative weight enhanced by his credentials as a board-certified clinical and anatomical pathologist.

On September 16, 1998, Dr. Caffrey rendered a consultative pathological report. He reviewed the autopsy report, 18 autopsy slides, the death certificate, as well as hospital and treatment records from 1985 through 1992. Dr. Caffrey conducted a microscopic examination and listed his findings under the headings of coronary arteries, heart, respiratory system, right lung, and left lung. Under respiratory system, Dr. Caffrey stated that slides E, F, G, and H were lung tissue that show similar changes of adhesions on the pleural surface with connective tissue and fat. He detected interstitial connective tissue proliferation. Throughout these slides, in the focal areas, Dr. Caffrey found a mild amount of anthracotic pigment, but he did not see typical macules and he found no nodules. He did not detect any evidence of progressive massive fibrosis. Under his review of the slides of the right lung, he found similar changes to the changes he noted under the respiratory system. He found the beginning formation of a granuloma with histiocytes. In all of the sections from the right lung, he only found a mild amount of anthracotic pigment in the focal areas. He did not find any macules, nodules, or complicated

pneumoconiosis. From his review of the left lung, Dr. Caffrey found, in the focal areas, pleural fibrosis with connective tissue and fat cells and a few mononuclear cells. Under this area, he found dilated alveoli with some interstitial connective tissue proliferation. Dr. Caffrey detected a significant degree of fibrosis in slide P without any true granulomas. He also found pleural fibrosis to be prominent in slide P, with a mild amount of anthracotic pigment present. He did not identify any macules or nodules. Under polarized light, he detected a few birefringent crystals, but he did not see changes consistent with silicosis. He diagnosed interstitial lung disease with idiopathic pulmonary fibrosis, chronic bronchitis, and a mild amount of anthracotic pigment in lung tissue. Dr. Caffrey then opined that he cannot make a diagnosis of CWP or any other occupational pneumoconiosis, including asbestosis. He opined that Miner suffered from an interstitial lung disease resulting in pulmonary fibrosis. He added that, "[m]ost always these diseases are idiopathic, by that I mean, cause unknown." Dr. Caffrey rendered extensive pathological findings and observations. He then concluded that Miner suffered from an interstitial lung diseases of unknown origin, but he ruled out CWP or any other occupational pneumoconiosis. However, he did not identify what evidence he relied upon that prevented him from diagnosing the presence of pneumoconiosis or any other occupational disease. Dr. Caffrey's opinion finding that Miner suffered from idiopathic pulmonary fibrosis is reasoned and documented. However, Dr. Caffrey's opinion on the absence of CWP is reasoned, but it is not documented because he failed to identify what microscopic findings he relied upon to determine that pneumoconiosis was not present. Therefore, I find that Dr. Caffrey's written opinion is entitled to a lesser degree of probative weight. However, in his deposition testimony, which is summarized below, Dr. Caffrey identified the minimum criteria for diagnosing the presence or absence of CWP. He identified the pathological findings and observations that he relied upon to conclude that simple CWP was not present. Therefore, I find that Dr. Caffrey, through a combination of his written report and his deposition testimony, has rendered a reasoned and documented opinion entitled to probative weight enhanced by his credentials as a board-certified clinical and anatomical pathologist.

Dr. Hutchins issued a consultative pathological report on September 30, 1998. He reviewed the autopsy report, 18 autopsy slides, and unidentified medical records. He stated that fourteen slides contain lung tissue, which show a pathologic process characterized by a mixed interstitial inflammatory cell infiltrate with mononuclear cells and plasma cells, interstitial granulomas of varying sizes as well as isolated Langhans type giant cells, interstitial fibrosis, and focal organizing pneumonia. He found parenchyma and scar type emphysema with bullous changes in some areas. Dr. Hutchins opined that the pathologic changes are typical of those found in chronic active extrinsic allergic alveolitis. He opined that extrinsic allergic alveolitis, in many instances, the result of hypersensitivity to an environmental antigen derived from fungi, animal proteins, or plants. From the available clinical and pathological information, Dr. Hutchins could not identify the cause of Miner's extrinsic allergic alveolitis. Dr. Hutchins also found that the slides show a slight to moderate amount of peri-vascular, peribronchial, and interstitial coal dust pigment with associated birefringent silicate-type particles. He did not find any coal macules, micronodules, macronodules, or lesions of progressive massive fibrosis. Thus, he opined that CWP was not present. He also found that there were no lesions of silicosis. Dr. Hutchins then concluded that Miner's pathological findings are those of a chronic active extrinsic allergic alveolitis, which would account for any pulmonary respiratory or disability that Miner had. He also found that, since it continued to be an active inflammatory reaction for ten

years after Miner's retirement from coal mine employment, it cannot be attributed to coal dust or other occupational exposure. Dr. Hutchins set forth pathological findings and observations, and his reasoning is supported by adequate data. His opinion is entitled to probative weight enhanced by his credentials as a board-certified anatomical pathologist.

Dr. Perper issued a narrative report on November 23, 1998. He reviewed Miner's autopsy slides, the consultative pathological reports of Drs. Naeye, Hutchins, and Caffrey, the autopsy report, and additional medical records. Dr. Perper rendered microscopic findings from the 18 slides. He found a marked distortion of pulmonary architecture due primarily to an interstitial pathological process and destructive alveolar changes. Dr. Perper documented marked thickening of pleura with fibrosis and slight to moderate anthracotic pigmentation and small numbers of birefringent silica crystals. He also documented marked thickening and fibrosis with marked chronic inflammation and anthracotic pigmentation, slight to moderate, and the presence of silica crystals in the pulmonary parenchyma and inter-alveolar and inter-lobular septa. He specifically noted that slide H showed a fibro-anthracotic area with chronic inflammation and numerous silica crystals with no evidence of multi-nucleated giant cells. He found slight to moderate clusters of anthracotic pigment with occasional silica crystals, both free and within macrophages, around blood vessels and bronchi as well as within alveolar spaces. In almost all of the nodularities he found, Dr. Perper detected slight to moderate anthracotic pigmentation with a few scattered birefringent silica crystals. Under polarized light, Dr. Perper detected many birefringent silica crystals scattered throughout the lymph nodes. Dr. Perper's microscopic diagnoses included a finding of CWP simple, with severe interstitial fibrosis and deposition of anthraco-silicotic deposits. Dr. Perper commented that he agreed with the findings of Dr. Dennis that CWP with the presence of macules, fibro-anthracotic-silicotic changes and associated interstitial fibrosis and centrilobular chronic emphysema were present. He also commented that the pneumoconiotic process had a definite interstitial pattern, with presence of silica crystal in the thickened septa with fibro-anthracosis and even within some of the silica crystals present in the interstitial granulomas and within the lymph nodes involved by the granulomatous reaction.

In support of his diagnosis of CWP, Dr. Perper identified scientific literature that he claimed substantiated that diffuse interstitial fibrosis ("DIF") of the lungs may be associated with non-asbestos pneumoconiosis, including silicosis and mixed-dust pneumoconiosis. Dr. Perper then opined that the opinions of the autopsy slides reviewers who denied the presence of pneumoconiosis were not convincing. He rendered specific criticisms of the opinions of Drs. Naeye, Hutchins, and Caffrey. He stated that Dr. Naeye minimized the presence of silica crystals and did not specifically diagnose the severe DIF as a non-asbestos pneumoconiosis. Dr. Perper disagreed with Dr. Naeye's opinion that the granulomatous disease present was sarcoidosis because he believed that it did not match the clinical or pathological characteristics of granulomatous process he identified in Miner. Dr. Perper criticized the opinion of Dr. Hutchins for denying the presence of CWP, including the denial of the presence of distinct macules, which was recognized by Drs. Dennis and Naeye and himself. He also criticized Dr. Hutchins for including his diagnosis of severe DIF as part of his diagnosis of allergic alveolitis, because he totally dismissed any significance to the presence of interstitial fibro-anthraco-silicosis, as well as his omission of any reference to the pattern of CWP associated with DIF. Dr. Perper also noted that Dr. Hutchins failed to diagnose the centrilobular emphysema that was even recognized

by Dr. Naeye. Dr. Perper criticized Dr. Caffrey's diagnosis of "interstitial lung disease with idiopathic pulmonary fibrosis" and mild anthracosis but denying the presence of CWP. He also criticized his failure to recognize the presence of centrilobular emphysema. Dr. Perper stated that it was difficult to understand Dr. Caffrey's opinion that the process was idiopathic (unknown cause) in the view of Miner's long occupational exposure to mixed coal containing silica and the presence of the silica crystals in Miner's lungs that Dr. Caffrey observed. Finally, he criticized Dr. Caffrey's failure to mention the possibility of CWP with a pattern of DIF.

Dr. Perper set forth pathological and clinical observations and findings, and his reasoning is supported by adequate data. He referenced scientific literature to support his conclusion. His opinion is reasoned and documented. I find that Dr. Perper's opinion is entitled to probative weight.

Dr. Kleinerman issued a consultative pathological report on May 12, 1999. He reviewed Miner's medical records, the autopsy report, and 18 autopsy slides. He rendered microscopic findings. Dr. Kleinerman stated that the microscopic sections of the lung show marked pleural fibrosis with subpleural nonspecific interstitial fibrosis and air space enlargement characteristic of Honeycomb lung. He noted that the adjacent lung tissue shows mild pulmonary edema with minimal black granular pigment in the areas of fibrosis. In the areas of lung parenchyma without fibrosis, Dr. Kleinerman found lesions of centriacinar emphysema. He noted findings around airspaces and bronchus that he found to be characteristic of chronic bronchitis. Dr. Kleinerman identified focal areas of acute bronchopneumonia. He found no evidence of simple CWP, simple silicosis, nor of complicated pneumoconiosis. In summary, Dr. Kleinerman stated that there appears to be extensive nonspecific interstitial fibrosis with honeycombing and evidence of organizing pneumonitis with granulation tissue. He found the pathological findings to be suggestive of two discrete and different pathologic processes which appear to be the cause of the interstitial fibrosis: (1) post pneumonic organizing pneumonitis and (2) sarcoidosis. Again, Dr. Kleinerman concluded that there is no evidence of simple CWP or silicosis. Dr. Kleinerman set forth pathological findings and observations. However, he failed to provide any documentation to support his conclusion that there is no evidence of the presence of CWP. There is adequate objective data to support his conclusion, but he failed to identify what documentation or lack thereof he relied upon to conclude that CWP was absent. This opinion is not properly documented. I find that Dr. Kleinerman's opinion is entitled to a lesser degree of probative weight.

On May 20, 1999, Dr. Kleinerman rendered a supplemental consultative report after reviewing Miner's medical records and the reports of Drs. Naeye, Hutchins, Caffrey, and Perper. Dr. Kleinerman stated that he strongly disagreed with Dr. Perper's conclusion that Miner had evidence of CWP. He referenced the *Archives of Pathology and Laboratory Medicine* (July 1979) as a scientific source that contains the criteria for diagnosing the presence of CWP. He stated that the required criteria is not present in Miner's lung tissue, so a diagnosis of simple CWP cannot be made or accepted. Dr. Kleinerman then referenced Dr. Perper's conclusion that DIF may be caused by coal mine dust and is present in Miner's lungs. He also noted the studies that Dr. Perper relied upon to support his conclusion regarding the etiology of Miner's DIF. Dr. Kleinerman opined that the studies relied upon by Dr. Perper provide no reliable basis to conclude that DIF occurs more frequently in a coal mining population than it does in a control

population due to his opinions that the three studies suffered from sampling bias and a failure to exclude other forms of recognized causes of DIF. Dr. Kleinerman then reiterates his opinion that Dr. Perper wrongly diagnosed CWP, interstitial fibrosis, and manifestations of restrictive lung disease and COPD since Miner clearly did not have CWP as documented by the absence of pathologic lesions of simple CWP in Miner's lung tissue sections. He determined that the amount of black granular pigment in Miner's lung tissue was minimal. Dr. Kleinerman then identified studies that he contended contradicts Dr. Perper's opinion that there are medical studies that establish that centrilobular (centriacinar) emphysema can be caused by exposure to coal mine dust or simple CWP. He stated that the studies relied upon by Dr. Perper do not control for cigarette smoking. Dr. Kleinerman identified focal emphysema as the only form of emphysema that is well known to be associated with coal dust inhalation. In conclusion, after reviewing the medical records and the literature referenced by Dr. Perper, Dr. Kleinerman opined that Dr. Perper's references do not provide a scientifically acceptable support for his conclusion that coal mine dust caused Miner's centrilobular emphysema. He also concluded that there is no pathology evidence that Miner has simple CWP. Thus, Dr. Kleinerman opined that none of Miner's associated clinical, physiological or other pathological findings are the result of simple CWP. Dr. Kleinerman set forth pathological findings and observations, and his reasoning is supported by adequate data. He relied on scientific literature to support his opinion. His opinion is reasoned and documented. I find that Dr. Kleinerman's supplemental opinion is entitled to probative weight enhanced by his credentials as a board-certified anatomical and clinical pathologist.

On September 27, 1999, Dr. Naeye rendered a second opinion after reviewing chest x-ray reports from 1998 and the pathological reports of Drs. Hutchins, Caffrey, and Perper. Dr. Naeye noted that Dr. Hutchins found the primary disease process in Miner's lungs to be an interstitial disease termed extrinsic allergic alveolitis. He stated that he observed the same disease process and described its location as both interstitial and micronodular. Because the disease he identified was sometimes micronodular, Dr. Naeye characterized it as sarcoidosis. However, he stated that there was no significant disagreement between his opinion and Dr. Hutchins opinion regarding the nature of the disease. Dr. Naeye also stated that he made the diagnosis of very mild, simple CWP because a few anthracotic macules were present in peribronchiolar locations that had some admixed fibrous tissue and a few non-toxic silicate crystals. He noted that Dr. Hutchins did not consider that these findings met the minimal criteria for the diagnosis of CWP. He also noted that Dr. Caffrey agreed with Dr. Hutchins on this point. Dr. Naeye then addressed Dr. Perper's report, noting that Dr. Perper implied that the interstitial process in Miner's lungs is a form of silicosis. Dr. Naeye criticized Dr. Perper for not distinguishing between silicates (which are non-toxic and easy to see) from free silica (toxic, very tiny and very difficult to see). He noted that he had reviewed the lungs of several thousand coal miners for the United States Department of Labor and had not once seen the interstitial process produced by free silica in the lungs of a bituminous miner. Dr. Naeye stated that exposures to high concentrations of free silica in the atmosphere are required to produce silicotic interstitial fibrosis. In such cases, he noted that myriads of very tiny birefringent crystals in the interstitial fibrosis are evident. Dr. Naeye concluded that the latter finding is not present in Miner's lungs.

Dr. Naeye was deposed on October 12, 1999. He testified that the question of whether coal dust inhalation causes centrilobular emphysema is the "$64,000.00 question" because it has

been a difficult issue over many years with scores of studies addressing the question. Dr. Naeye answered that centrilobular emphysema is not caused by coal dust inhalation. He testified that coal dust in some locations of the world causes centrilobular emphysema, such as in Great Britain. Dr. Naeye testified that when the hard and intermediate grade coal was mined in Pennsylvania, it caused terrible lung disease in the form of fibrosis and centrilobular emphysema. The further south into West Virginia and the Appalachian chain, he stated that the coal dust is relatively innocuous, only causing chronic bronchitis and not centrilobular emphysema. Dr. Naeye described interstitial fibrosis as a terrible disease that selectively destroys the walls of the air spaces, the alveoli walls and it spreads over a large part of the lungs. He added that a few of the causes of the infection are known, but very often (85% of the time) the source of the viral infection that started the process is not known. Dr. Naeye identified the minimum criteria for diagnosing CWP by autopsy evidence is the presence of black macules that are small round macules of at least one millimeter in diameter. If all of the black macules are smaller than one millimeter, they have to have associated fibrosis and a realm of emphysema called focal emphysema (which is different than centrilobular emphysema). Dr. Naeye testified that there has to be fibrous tissue mixed with the black pigment and realms of focal emphysema around the black deposits. He added that if there are only black deposits and no fibrosis, no matter how much black pigment deposition there is, no malfunction of the lung will be caused. From the 13 slides of lung tissue, Dr. Naeye commented that he received enough lung tissue to make or exclude a diagnosis of CWP. He testified that he was not at any disadvantage by not doing the actual autopsy because Miner had lots of studies. Dr. Naeye added that if it is hard to decide whether simple CWP is present or absent, he gives the benefit of the doubt to the coal miner. He testified that he made the diagnosis of simple CWP because there were lesions that he felt met the minimum criteria. He found the lesions to be extremely insignificant by comparison to the major disease Miner had in his lungs. Dr. Naeye reiterated his finding that Miner had a granulomatous interstitial process that destroyed the walls of the air spaces. He noted that the process doesn't look anything like CWP. Dr. Naeye reiterated the criticisms of Dr. Perper's report that he levied in his May 20, 1999 report. Dr. Naeye also criticized Dr. Dennis' opinion that coal dust or anthracotic silicotic pigment deposition caused Miner's interstitial pulmonary fibrosis because the pattern of interstitial fibrosis that is caused by exposure to free silica in coal mining was a completely different pattern than the pattern present in Miner's lungs.

Dr. Caffrey was deposed on October 29, 1999. Dr. Caffrey identified the minimum criteria for diagnosis the presence of CWP by autopsy evidence. He opined that he was not at a disadvantage to the autopsy prosector because the prosector is trained to take appropriate sections to establish that pathologists reviewing the slides are on a level playing field. Dr. Caffrey testified that the abnormalities he observed in the slides of Miner's lung tissue were not related to coal dust inhalation. He reiterated his conclusion that Miner suffered from idiopathic pulmonary fibrosis. He noted that the exact cause of the pulmonary fibrosis is not known, and he added that some experts have identified as many as 150 causes of interstitial pulmonary fibrosis. Regarding his finding of the presence of a mild amount of anthracotic pigment in the lung, Dr. Caffrey testified that it was only a mild amount and it did not stimulate the production of reticulin and there was no focal emphysema, so Miner did not have the minimum criteria to diagnose the presence of the lesion of CWP. He disagreed with Dr. Dennis' respiratory system diagnosis because he did not identify macules because coal dust did not stimulate the production

of reticulin and there was no focal emphysema. He also added that he has no idea what Dr. Dennis meant by papules because that is not a term he uses in pulmonary pathology.

Drs. Naeye, Caffrey, and Kleinerman relied upon the same scientific source to identify the minimum criteria for diagnosing the presence of simple CWP. Dr. Naeye phrased the minimum criteria as a finding of a few small black deposits adjacent to small airways and arteries with some admixed fibrous tissue and a few birefringent crystals). From his macroscopic analysis, Dr. Dennis found the presence of black macules with pigment deposition varying in size from .3 to .5 centimeters, with sections through the pulmonary architecture showing emphysematous changes, black pigment deposition, and black pigment surrounding major vessels and bronchi. Under microscopic analysis, Dr. Dennis detected the presence of black pigment showing birefringent crystalline structures compatible with silicosis, as well as nodules varying in size from .1 to .5 millimeters. Dr. Dennis also found, during microscopic examination, markedly thickened pleura, silicotic and anthracotic nodules associated with the collapse of septal walls and subsequent collapse of alveolar septal spaces, and broadened fibrous tissue septi along with a granulomatous response. His final diagnosis included a finding of anthracosilicotic pigment deposition and pleural reaction, moderate to severe, with extreme pulmonary fibrosis and restrictive lung disease with features related to anthracosilicosis. Similar to Dr. Dennis, but to a lesser extent, Dr. Naeye found the minimum findings for diagnosing simple CWP were present because he found fibrous tissue associated with black pigment as well as rare admixed small and medium-sized birefringent crystals. In his deposition testimony, Dr. Naeye reiterated his conclusion that Miner's lungs contained the minimum findings for diagnosing the presence of pneumoconiosis. While he testified that he gives coal miners the benefit of the doubt when it is difficult to determine whether the minimum findings required to diagnose CWP are present, his conclusion that CWP was based on his actual findings and not a presumption. Dr. Perper found the presence of simple CWP as well. He found evidence of fibrosis and slight to moderate anthracotic pigmentation and small numbers of birefringent silica crystals. I accorded an enhanced degree of probative weight to the opinion of Dr. Dennis based on his role as the autopsy prosector because he identified that he was only able to extricate a limited amount of tissue without tearing the lung tissue. Drs. Naeye and Caffrey both testified that they were not at a disadvantage to Dr. Dennis, but did not address Dr. Dennis' statement regarding his statements that the marked fibrosis present prevented complete removal of lung tissue. Dr. Dennis, through his position as the autopsy prosector, was in a better position to evaluate the extent and nature of the disease process contained in Miner's lungs. His observations, especially his macroscopic findings, were the benefit of viewing Miner's entire respiratory system. The other reviewing pathologists had to base their opinions on limited lung sections. Dr. Dennis' finding of black macules with pigment deposition varying in size from .3 to .5 centimeters in diameter were contained in his macroscopic description. Dr. Perper's opinion is reasoned and documented and entitled to probative weight. I accorded an enhanced degree of probative weight to the opinion of Dr. Naeye because his opinion was reasoned and documented and because he is board-certified in clinical and anatomical pathology.

Drs. Kleinerman, Caffrey, and Hutchins opined that the minimum criteria to diagnose the presence of CWP was not present. Dr. Hutchins found a slight to moderate amount of peri-vascular, peri-bronchial, and interstitial coal dust pigment with associated birefringent silicate-type crystals, but he did not find macules, micronodules, macronodules, or lesions of progressive

massive fibrosis. Dr. Caffrey found a mild amount of anthracotic pigment, but he too did not see typical macules or nodules. In one slide, Dr. Caffrey found significant fibrosis with a mild amount of anthracotic pigment, but no macules or nodules. Dr. Kleinerman found lung tissue showing mild pulmonary edema with minimal black granular pigment in the areas of fibrosis. I accorded Dr. Hutchins opinion an enhanced degree of probative weight since his opinion was reasoned and documented and he was a board-certified anatomical pathologist. I also accorded Dr. Caffrey's reasoned and documented opinion enhanced probative weight based on his board-certification in clinical and anatomical pathology. However, I accorded a lesser degree of probative weight to the opinion of Dr. Kleinerman because he failed to identify what evidence he relied upon to support his conclusion that simple CWP was not present. Dr. Kleinerman did not identify what he considered the minimum findings of simple CWP.

I find that Claimant has established, by a preponderance of the autopsy evidence, that Miner suffered from coal workers' pneumoconiosis at the time of his death. The probative weight accorded to the opinions of Drs. Dennis, Perper, and Naeye are sufficient to outweigh the contrary opinions of Drs. Caffrey, Kleinerman, and Hutchins.

Dr. Perper also opined that Miner's interstitial pulmonary fibrosis was caused by coal dust inhalation. He relied upon medical literature and defended his opinion in the face of criticism from other pathologists of record. Drs. Caffrey and Naeye strongly criticized the opinion of Dr. Perper. Dr. Naeye's report and deposition testimony constitutes the most compelling evidence regarding the etiology of Miner's interstitial pulmonary fibrosis because of this experience in evaluating lung diseases suffered by coal miners and his analysis of the role that the hardness of the coal dust plays in causing interstitial pulmonary fibrosis. I find that the opinions of Drs. Naeye, Hutchins, Kleinerman, and Caffrey establish that Miner's interstitial pulmonary fibrosis is idiopathic, and that coal dust inhalation was not a contributing cause of Miner's interstitial pulmonary fibrosis. There is no medical evidence that establishes a finding of the presence of idiopathic pulmonary fibrosis to be mutually exclusive with a finding of CWP. In fact, Dr. Naeye diagnosed the presence of an interstitial pulmonary fibrosis of unknown etiology and the presence of mild, simple CWP. I find that the evidence establishes that Miner suffered from coal workers' pneumoconiosis and idiopathic interstitial pulmonary fibrosis at the time of his death. Therefore, I find that the Claimant has established the existence of pneumoconiosis through autopsy evidence under subsection (a)(2).

(3) Section 718.202(a)(3) provides that pneumoconiosis may be established if any one of several cited presumptions are found to be applicable. In this case, the presumption of § 718.304 does not apply because there is no evidence in the record of complicated pneumoconiosis. Section 718.305 is not applicable to claims filed after January 1, 1982. Finally, the presumption of § 718.306 is applicable only in a survivor's claim filed prior to June 30, 1982. Therefore, Claimant cannot establish pneumoconiosis under subsection (a)(3).

(4) The fourth and final way in which it is possible to establish the existence of pneumoconiosis under § 718.202 is set forth in subsection (a)(4) which provides in pertinent part:

> A determination of the existence of pneumoconiosis may also be made if a physician, exercising sound medical judgment, notwithstanding a negative x-ray, finds that the miner suffers or suffered from pneumoconiosis as defined in § 718.201. Any such finding shall be based on electrocardiograms, pulmonary function studies, physical performance tests, physical examination, and medical and work histories. Such a finding shall be supported by a reasoned medical opinion.

§ 718.202(a)(4).

This section requires a weighing of all relevant medical evidence to ascertain whether or not the claimant has established the presence of pneumoconiosis by a preponderance of the evidence. Any finding of pneumoconiosis under § 718.202(a)(4) must be based upon objective medical evidence and also be supported by a reasoned medical opinion. A reasoned opinion is one which contains underlying documentation adequate to support the physician's conclusions. *Fields v. Island Creek Coal Co.*, 10 B.L.R. 1-19, 1-22 (1987). Proper documentation exists where the physician sets forth the clinical findings, observations, facts, and other data on which he bases his diagnosis. *Oggero v. Director, OWCP*, 7 B.L.R. 1-860 (1985).

The physicians who rendered narrative opinions on the question of whether or not Miner suffered from pneumoconiosis at the time of his death stated that autopsy evidence is the most reliable method for determining the presence or absence of pneumoconiosis. The Board has also recognized that autopsy evidence is the most reliable evidence of the existence of pneumoconiosis. *Terlip v. Director, OWCP*, 8 B.L.R. 1-363 (1985). Thus, I find that the most probative narrative medical opinions would be those opinions rendered after the physician considered Miner's autopsy findings. The record does not contain a narrative opinion based on evidence outside of the autopsy findings that is more probative of Miner's condition at the time of his death. The weight of the autopsy evidence establishes the presence of pneumoconiosis. Therefore, I find it unnecessary to discuss whether the weight of the evidence establishes the presence or absence of pneumoconiosis under subsection (a)(4).

Arising Out of Coal Mine Employment

In order to be eligible for benefits under the Act, Claimant must also prove that Miner's pneumoconiosis arose, at least in part, out of his coal mine employment. § 718.203(a). For a miner who suffers from pneumoconiosis and was employed for ten or more years in one or more coal mines, it is presumed that his pneumoconiosis arose out of his coal mine employment. *Id.* As I have found that Miner engaged in 11 years of coal mine employment, and as no rebuttal evidence was presented, I find that Claimant's pneumoconiosis arose out of his coal mine employment in accordance with the rebuttable presumption set forth in § 718.203(b).

Death Due to Pneumoconiosis

Mrs. Thacker has established, by a preponderance of the chest x-ray and autopsy evidence, that Miner suffered from pneumoconiosis arising out of coal mine employment. She must now prove that Miner's death was due to pneumoconiosis in order to be entitled to benefits.

Subsection 718.205(c) applies to survivor's claims filed on or after January 1, 1982 and provides that an eligible survivor will be entitled to benefits if any of the following criteria are met:

1. Where competent medical evidence establishes that pneumoconiosis was the cause of the Miner's death, or

2. Where pneumoconiosis was a substantially contributing cause or factor leading to the miner's death or where death was caused by complications of pneumoconiosis, or

3. Where the presumption set forth in § 718.304 (evidence of complicated pneumoconiosis) is applicable.

20 C.F.R. § 718.205(c).

Pneumoconiosis is a "substantially contributing cause" of a miner's death if it hastens the miner's death. § 718.205(c)(5). The presumption set forth in § 718.304 is not applicable because Claimant has not established the presence of complicated pneumoconiosis. Therefore, in order for Claimant to be entitled to benefits, she must show that pneumoconiosis was the direct cause of Miner's death or that pneumoconiosis hastened Miner's death.

A death certificate, in and of itself, is an unreliable report of the miner's condition and it is error for an administrative law judge to accept conclusions contained in such a certificate where the record provides no indication that the individual signing the death certificate possessed any relevant qualifications or personal knowledge of the miner from which to assess the cause of death. *Smith v. Camco Mining, Inc.*. 13 B.L.R. 1-17 (1989); *Addison v. Director, OWCP*, 11 B.L.R. 1-68 (1988).

Dr. Nichols completed and signed Miner's death certificate on April 14, 1998. Dr. Nichols listed the date of death as April 6, 1998. He did not mark whether or not he had reviewed the autopsy findings before he completed the death certificate. Dr. Nichols listed the immediate cause of death as respiratory failure due to or as a consequence of the underlying cause of pulmonary fibrosis. Dr. Nichols admitted Miner to Pikeville Methodist Hospital on March 8, 1998, and he attended to Miner until Miner died on April 6, 1998. He rendered final diagnoses of pulmonary fibrosis with expiratory failure, methicillin resistant pneumonia, and congestive heart failure. From his previous treatment of Miner, Dr. Nichols noted that Miner had a long-standing history of pulmonary fibrosis. When he admitted Miner to the hospital, Miner was experiencing severe hypoxemia. He added that Miner had been using continuous supplemental oxygen, and had been sick for three weeks prior to admission. Dr. Nichols noted that Miner had a progressively down-hill hospital course leading to his death on April 6, 1998. Dr. Nichols commented that Miner required multiple antibiotics to eradicate his pneumonia. Miner became dehydrated from his tube feedings, which caused him to go into mild heart failure.

When attempts were made to correct Miner's heart failure, he expired. Dr. Nichols possessed sufficient knowledge of Miner's condition from which to assess the cause of death.[3] He followed Miner for the last month of his life, and had previously treated Miner for respiratory disease. I accord probative weight to the cause of death listed on the death certificate.

Dr. Dennis conducted the autopsy. He found the presence of CWP and interstitial fibrosis and granulomatous inflammation. Dr. Dennis opined that Miner died a pulmonary death. He asserted that Miner's pathology was moderate to severe. Dr. Dennis stated that the etiological factors of Miner's pathology were coal dust or anthracosilicotic pigment deposition with resultant fibrosis and pulmonary reactions secondary to the process. His autopsy report complied with the quality standards of § 718.106. He set forth macroscopic findings and microscopic findings. Dr. Dennis did not review any of Miner's medical records, and there is nothing in his report to indicate that his report was based on any evidence besides his autopsy findings. Dr. Dennis' opinion that Miner suffers from CWP and that he died a pulmonary death is supported by his pathological findings and observations. However, his report does not clearly identify what was the cause of the pulmonary death, nor does he specify what role CWP played, if any, in contributing to Miner's death. The weight attributed to Dr. Dennis' report regarding the cause of Miner's death must be limited to his finding that Miner died a pulmonary death.

In his August 8, 1998 report, Dr. Naeye reviewed and summarized the autopsy report, 18 autopsy slides, and Miner's hospital records. He opined that a widespread granulomatous process, much of which he found in an interstitial location, caused maximum disability and led to Miner's death. While Dr. Naeye did find the minimum findings required to diagnose simple CWP, he determined that the CWP actually present was far too mild to have caused any abnormalities in lung function that would have played any role in Miner's death. Dr. Naeye concluded that neither CWP nor any other occupational disorder contributed to Miner's death. On September 27, 1999, Dr. Naeye rendered a supplemental opinion based on his review of the reports of Drs. Hutchins, Caffrey, and Perper. He also reviewed additional chest x-ray interpretations. Dr. Naeye noted that Drs. Caffrey and Hutchins disagreed with his opinion that the minimum findings for diagnosing CWP were present. However, he stated that he agreed with Drs. Hutchins and Caffrey that neither Miner's CWP nor any other occupational lung disease has any role in causing or hastening Miner's death.

Dr. Naeye was deposed on October 12, 1999. He reiterated his finding that he detected the minimum findings necessary to diagnose the presence of simple CWP. Dr. Naeye also reiterated his concurrence with the findings of Drs. Kleinerman and Caffrey that Miner's interstitial pulmonary fibrosis was not caused or contributed to by Miner's coal dust exposure.

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[3] Dr. Nichols received consultative reports from other physicians in 1990, 1992, 1996, 1997, and 1998. He had previously admitted and attended to Miner at Pikeville Methodist Hospital on the following occasions: (1) December 14, 1996 through December 20, 1996 for COPD, pneumonia, and suspected asbestosis; (2) April 14, 1996 through April 23, 1996 for exacerbation of shortness of breath with a history of severe pulmonary fibrosis and suspected asbestosis; and (3) April 1, 1996 through April 5, 1996 for hypoxemia with notations that Miner had a history of limited lung capacity, black lung, and chronic lung disease. In 1989, Dr. Nichols authored a letter noting that he first examined Miner on June 26, 1980. He then examined Miner for the second time on March 25, 1987. He opined, on September 13, 1989 that he considered Miner a pulmonary cripple who must have developed an occupational disease, probably asbestosis, even though the diagnosis has not been proven.

He noted that the amount of CWP that he detected was so minimal that it had nothing to do with 99.9% of what caused Miner's pulmonary troubles. Dr. Naeye then testified:

> Oh, sure. In other words, it's so minimal. I guess people who aren't looking at these cases day in and day out the way I've had to do for so many years for the Department of Labor. If they saw this, coal workers' pneumoconiosis would probably never cross their minds because there's this other overwhelming dominating disease process present and most pathologists don't like to make two diagnoses if 99 percent of what's around can be explained by a single diagnosis.

Dr. Naeye then testified that Miner's CWP was so minor that it did not have any effect on lung function whatsoever. He added that Miner's CWP did not cause one-thousandth of one percent of disability, nor did it contribute in any way to Miner's outcome. Dr. Naeye opined that Miner's death was caused by interstitial pulmonary fibrosis. Dr. Naeye set forth pathological and clinical observations and findings, and his reasoning is supported by adequate data. Dr. Naeye's opinion was predicated on a finding of minimal, simple CWP arising out of coal mine employment. His opinion is reasoned and documented. I find that Dr. Naeye's opinion is entitled to probative weight enhanced by his credentials as a board-certified clinical and anatomical pathologist.

Dr. Caffrey issued a report on September 16, 1998. He reviewed the autopsy report, the autopsy slides, Miner's medical records, and the death certificate. However, the medical records he reviewed and summarized did not include any records from 1992 through Miner's death. From his review of the autopsy slides, he opined that Miner suffered from idiopathic interstitial pulmonary fibrosis, chronic bronchitis, and a mild amount of anthracotic pigment in the lung tissue. Dr. Caffrey concluded that he could not make a diagnosis of CWP or any other occupational lung disease. He referenced scientific literature that discussed the symptoms of idiopathic pulmonary fibrosis, which notes that the major cause of death is respiratory insufficiency. Dr. Caffrey noted that the death certificate listed the cause of Miner's death as respiratory failure secondary to pulmonary fibrosis, which he found to be consistent with what he found. Dr. Caffrey then concluded that Miner's occupation as a coal miner did not cause, contribute, or hasten his death. Dr. Caffrey was deposed on October 29, 1999. He reiterated the findings and conclusions contained in his report. Dr. Caffrey testified that there was no evidence of CWP. He opined that Miner died of respiratory failure secondary to idiopathic pulmonary fibrosis in concurrence with the death certificate. Dr. Caffrey's opinion on the etiology of Miner's death is based on the predicate that there was no evidence of simple CWP, which is contradictory to the undersigned's determination that Miner suffered from simple CWP at the time of his death. Therefore, I accord a lesser degree of probative weight to the opinion of Dr. Caffrey.

Similar to the opinion of Dr. Caffrey, Dr. Hutchins also opined that Miner did not suffer from CWP at the time of his death based on the autopsy findings. Dr. Hutchins opinion on the etiology of Miner's death is based on the predicate that there was no evidence of simple CWP, which is contradictory to the undersigned's determination that Miner suffered from simple CWP at the time of his death. Therefore, I accord a lesser degree of probative weight to the opinion of Dr. Hutchins.

Dr. Perper issued a report on November 23, 1998. He reviewed the autopsy report, autopsy slides, Miner's medical records, and the reports of Drs. Naeye, Hutchins, and Caffrey. From his review of the autopsy slides, Dr. Perper diagnosed the presence of CWP interstitial fibrosis and granulomatosis, emphysema, and pneumonia. He diagnosed pneumoconiosis by the presence of radiological evidence, autopsy evidence of macules and nodules, and he also found that Miner's coal dust inhalation contributed to Miner's DIF. Dr. Perper concluded that Miner's CWP and exposure to coal mine dust and silica was a substantial cause of Miner's death. He also found that there is unanimous convergence of clinical evidence, pathological evidence and opinions of all treating physicians, prosectors and medical consultants that Miner suffered from a severe, chronic restrictive and obstructive lung disease that was totally and permanently disabling and ultimately caused Miner's death. The undersigned determined, in the analysis conducted under § 718.202(a)(2), that coal dust inhalation was not an etiological factor of Miner's idiopathic pulmonary fibrosis. The undersigned found that the preponderance of the autopsy evidence established that Miner suffered from simple CWP. Dr. Perper opined that pneumoconiosis contributed to Miner's death and he identified that general evidence he relied upon, but he did not provide any detailed rationale to identify how Miner's simple CWP contributed to Miner's death. Since Dr. Perper failed to identify how Miner's simple CWP contributed to Miner's death, his report does not constitute a reasoned and documented opinion. I accord a lesser degree of probative weight to Dr. Perper's opinion.

Dr. Kleinerman rendered an opinion on May 12, 1999. He reviewed and summarized the autopsy report, autopsy slides, and Miner's medical records. Dr. Kleinerman conducted a microscopic analysis of the autopsy slides and concluded that there was no evidence of simple CWP or silicosis. Dr. Kleinerman also concluded that Miner's coal mine dust exposure was not a contributing factor of some discernible consequence to Miner's disabling respiratory impairment. Finally, Dr. Kleinerman stated that Miner's death was not caused by or hastened by coal mine dust or CWP. Dr. Kleinerman did not identify what caused Miner's death. He did not set forth any rationale to explain how he determined that CWP did not cause or contribute to Miner's death. Presumably, since he found that Miner did not suffer from CWP, he opined that Miner's death was not due to CWP since he found that Miner did not suffer from CWP. In a supplemental report rendered on May 20, 1999, Dr. Kleinerman reviewed the reports of Drs. Naeye, Caffrey, Hutchins, and Perper. He opined that there was no pathological evidence of simple CWP. Since CWP was not present, Dr. Kleinerman opined that CWP cannot be considered a substantial cause of Miner's death. The undersigned determined that Claimant established that Miner suffered from CWP at the time of his death. Dr. Kleinerman's opinion that CWP did not cause or contribute to Miner's death is predicated on the erroneous finding that Miner did not suffer from CWP. Therefore, I accord a lesser degree of probative weight to Dr. Kleinerman's opinion.

I find that Claimant has failed to establish, by a preponderance of the evidence, that Miner's death was due to pneumoconiosis arising out of coal mine employment. Dr. Naeye's opinion is the most probative opinion on this issue because he considered the presence of simple CWP and determined that Miner also suffered from idiopathic pulmonary fibrosis. Dr. Naeye determined that Miner's CWP was clinically insignificant and found that Miner's death was caused by respiratory failure secondary to idiopathic pulmonary fibrosis. Dr. Naeye's reasoned and documented opinion is better supported by the objective evidence than the opinion of Drs.

Perper, Hutchins, Caffrey, Kleinerman, and Dennis. Dr. Naeye testified about his extensive expertise in evaluating the lungs of coal miners, especially regarding determining the etiology of pulmonary fibrosis. Based on the strength of Dr. Naeye's credentials and the reasoning he employed, I accord his opinion controlling weight. The record does not contain a reasoned and documented medical opinion finding that Miner's death was due to pneumoconiosis. Thus, I find that Miner's death was not caused, contributed to, or hastened by his pneumoconiosis arising out of coal mine employment.

Entitlement

Claimant, Geraldine Thacker, has failed to prove, by a preponderance of the evidence, that Miner's death was due to pneumoconiosis. Therefore, Mrs. Thacker is not entitled to benefits under the Act.

Attorney's Fees

An award of attorney's fees is permitted only in cases in which the claimant is found to be entitled to benefits under the Act. Because benefits are not awarded in this case, the Act prohibits the charging of any fee to the Claimant for the representation and services rendered in pursuit of the claim.

ORDER

IT IS ORDERED that the claim of Geraldine Thacker for benefits under the Act is hereby DENIED.

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THOMAS F. PHALEN, JR.
Administrative Law Judge

NOTICE OF APPEAL RIGHTS

Pursuant to 20 C.F.R. § 725.481, any party dissatisfied with this Decision and Order may appeal it to the Benefits Review Board within 30 days from the date of this decision, by filing notice of appeal with the Benefits Review Board, P.O. Box 37601, Washington, D.C. 20013-7601. **A copy of a notice of appeal must also be served on Donald S. Shire, Esquire, Associate Solicitor for Black Lung Benefits, Frances Perkins Building, Room N-2117, 200 Constitution Avenue, NW, Washington, D.C. 20210.**